

# Susceptibility to Auditory Fatigue

W. DIXON WARD

HEARING RESEARCH LABORATORY,  
 DEPARTMENT OF OTOLARYNGOLOGY,  
 UNIVERSITY OF MINNESOTA,  
 MINNEAPOLIS, MINNESOTA

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## I. INTRODUCTION

### A. Historical Aspects

It has long been realized that individuals differ widely in their ability to stay healthy. Some persons display cold symptoms nearly constantly; others never do. Exposed to a virulent disease, some organisms contract it and die; others are only briefly ill. Bombarded by equal amounts of radiation, some animals succumb several times as swiftly as others. The general problem of individual differences in susceptibility to noxious stimuli was last discussed in a comprehensive fashion by Williams (1956).

Such variability between individuals is also found in hearing loss due to noise. Although the deafness found in metalworkers has probably been recognized as a clinical entity for several millenia, there no doubt has also been an awareness that some smiths become deafer than others. In 1830-1831, for example, Foscroke wrote: "In the absence of hereditary predisposition, as indeed in the majority of those who go deaf, there would appear to be some original condition of the organs, which renders them in a particular degree susceptible of being acted upon by the exciting causes. Otherwise, why should it occur that under all the same conditions of the case, the same remote causes should produce it not in one individual immediately excite it in another? This original condition consists probably in some original imperfection in the constitution of the ear in structure and function."

There is no doubt that Foscroke's empirical facts were correct enough: in a group of people exposed to the same average working conditions, some will indeed lose hearing faster than others, so that if these susceptible persons could be kept out of noisy jobs, society and the workers themselves would all benefit. There are, however, at least three possible hypotheses that would have adequately explained the facts available to Foscroke. Let me state them in an extreme form, in order to make them mutually exclusive.

Hypothesis I: *Fixed susceptibility*. Because of certain inherent physiological properties of its hearing mechanism, each organism possesses a distinct, if not unique, susceptibility to damage; thus a given acoustic event will inevitably produce a particular result on each ear.

Hypothesis II: *Variable susceptibility*. All organisms are, structurally speaking, actually equally resistant to damage *on the average*, but because of changes in the internal physiological milieu associated with the ongoing activity of the organism, this susceptibility varies from instant to instant, from minute to minute, and from day to day; thus a given

acoustic event will produce the most hearing loss in those who happen to have unusually high susceptibility at the time.

Hypothesis III: *Variable exposure*. All ears are much alike in susceptibility, but because of the fact that two objects cannot occupy the same space at the same time, no two organisms will be given exactly the same exposure; variability in the effects of a particular environment can therefore be completely attributed to actual differences in the amount of sound reaching the ears of different workers. Thus, the presence of a slight hearing loss in a worker does not necessarily mean he is unusually susceptible—he may merely have been exceptionally unlucky.

Foscroke, it can be seen, championed the fixed-susceptibility theory. Not only that, but his statement that high susceptibility is due to "some original imperfection" implies that there is a clear distinction between normal and abnormal susceptibility. No doubt what Foscroke had in mind was a specific weakness like hemophilia—one either "has" it or he doesn't. Also implicit in Foscroke's statement is the proposition that the ear most susceptible to one type of acoustic stimulation will also be more susceptible to all others.

Enough research has been performed since Foscroke's time to prove that no single one of these alternatives is the whole story, but that all three are partially true. Even when the exposures are rigidly controlled and matched, there is a large range of effects (Miller *et al.*, 1963), so exposure variability is not the sole basis of observed differences between organisms. Furthermore, these differences are continuously distributed, so we can safely abandon the supposition that the distribution is dichotomous. We may speak of *high* and of *low* susceptibility, but not of "susceptible" against "nonsusceptible" ears.

### B. TTS as a Susceptibility Index

Now the main difficulty in measuring susceptibility directly—and an insuperable one—is that once an ear has been damaged, it is no longer the same ear. We cannot test the proposition that an ear more susceptible to one type of noise will also be more susceptible to all others, nor can we determine the magnitude of day-to-day variability in susceptibility. Besides, it would be at least absurd, if not worse, to establish that a man is susceptible to hearing loss by giving him one.

For the last 40 years, therefore, considerable effort has been expended in an effort to find a swift, *reversible* test for predicting susceptibility. Since any factor that affects the transmission of sound to the cochlea must be involved, one might attempt to measure such things as differences in the mass and geometry of the ossicles, the area of the tympanic membrane and the oval window, the elasticity of the skin in the external

and middle ears, and the strength and size of the middle-ear muscles; indeed, even the pneumatization of the mastoid seems to be involved in susceptibility (Link and Handl, 1955). However, also necessarily contributing to differences in susceptibility are variables that we cannot observe directly, such as differences in the stiffness of the cochlear partition, density and spacing of the hair cells, thickness of the tectorial membrane, adequacy of the blood supply to the cochlea, chemical characteristics of the endolymph, oxygen metabolism, and density of afferent and efferent innervation of the hair cells.

In the face of such complexity, it was clear to the early investigators that the best chance to predict susceptibility to *permanent* loss probably was embodied in tests in which *temporary* (reversible) losses were deliberately produced by acoustic exposures either shorter or less intense than exposures that would produce permanent loss. Until the development of electronic audiometric equipment in the late 1920's, the measurement of the temporary threshold shift (TTS) produced by a given exposure was difficult—indeed, guaranteeing the constancy of the TTS-producing tone from listener to listener was no mean task. From Urbantschitsch's pioneering experiments on TTS in 1881, until 1930, when Peyser first attempted to use differences in TTS produced in different individuals as an index of susceptibility, the only measure of TTS was the shortening of the duration of perception of a tuning fork struck in a standard manner. The development of reasonably reliable TTS tests is therefore a very recent endeavor.

The main advantage of TTS as a predictor of susceptibility is that it will depend on much the same list of variables as those we assume to be responsible for differences in permanent threshold shifts (PTS): the physiological structures determining the energy reaching the cochlea, characteristics of the basilar membrane, oxygen metabolism, and so on. It is thus not at all unreasonable to advance the hypothesis that the ear showing the most TTS from a short exposure to a noise will show the most PTS after a long exposure. [A more detailed discussion of susceptibility tests and their implication has been presented elsewhere (Ward, 1965b).]

Beginning with Peyser in 1940, many different TTS tests have been proposed as susceptibility indices. They are listed in Table I. The early tests involved exposures to pure tones from the audiometer, TTS being measured at the same frequency as the fatiguing stimulus. With the growth of knowledge about TTS, it became clear that for reasonably intense exposures, the maximum effect occurred half an octave above the exposure frequency (Theilgaard, 1949), and so the TTS was later measured there. Likewise, the use of low-frequency tones as fatiguing

TABLE I  
Proposed Susceptibility Tests Involving Temporary Threshold Shifts

Report	Stimulus (kc)	Level (db)	Duration (min)	Recovery time (min)	Test frequency (kc)
Peyser (1940)	0.25	80 (HL)	0.5	0.5	0.25
Wilson (1943)	0.25	80 HL	5	1	Octaves of 0.25
Peyser (1943)	1	100 (HL)	3	0.25	1
Theilgaard (1949)	0.5, 1, 2, 4	100 HL	5	5	Half-octave above exposure
Theilgaard (1951)	1	100 HL	5	5	1.5
Tanner (1955)	1	100 HL	5	5	1
Theilgaard, according to Greisen* (1951)	1.5	100 HL	5	5	2
Wilson (1944)	2	80 HL	8	1	Octaves of 0.25
Harris (1954)	2	97 SPL	5	2	4
Palva (1958)	2	30 SL	2	2	2
van Dishoeck (1956)	2.5	100 (HL)	3	0.25	All (sweep)
Greisen (1951)	3	80 and 90 HL	5	5	4
Jørgen and Carhart (1955)	3	105 SPL	1	Parameter	4.5
Jørgen and Carhart (1956)	3	100 SPL	1	Parameter	2, 4, 6
Wheeler (1950)	Noise	105 SPL	30	Parameter	2, 4, 6
Gallagher and Goodwin (1952)	Noise	115 HL	10	"Immediately"	4
Rüdel (1954)	Noise	Parameter	2	4	3
Falconnet et al. (1955)	Noise	100 SPL	3	Parameter	0.5, 15
Christiansen (1956)	Noise	105 (HL)	3		4

\* "115 db above normal threshold: 6 milliwatts across the phone" (which was a PDR-10).

\* Creisen says Theilgaard used 1500 cps as a fatiguer, but Theilgaard's published reports indicate only 1000 cps.

\* SPL = sound pressure level (db re 0.0002 dyne/cm<sup>2</sup> rms pressure). SL = sensation level (db above the individual listener's threshold). HL = hearing level (audiometer dial). Parentheses indicate that the article merely stated "db" (no reference level given).

stimuli was gradually abandoned, mainly because more TTS was produced by high than by low frequencies.

Pure tones were first used as fatiguing stimuli primarily because they were handy—all the tester had to do to expose an ear was to flip the dial on his audiometer up to the maximum, for the required time. Wheeler (1950) argued that white noise should be used as a fatiguing stimulus, not only because it is more realistic—i.e., more like the sounds of industry—but because it would produce a TTS over a broad range of frequencies instead of just at and above the exposure frequency.

The usual procedure for TTS tests involved measurement of threshold at the test frequency (Table 1, column 6), exposure to a stimulus (column 2) at a set level (column 3) for a fixed time (column 4), and finally, after a more or less constant delay (column 5), determination of the shifted threshold at the test frequency. The term "parameter" in column 5 indicates that the shifted threshold was measured at several times, so that the course of recovery might be followed. The reasoning here was that not only the initial TTS, but also the rate of recovery from this shift, is important in the determination of susceptibility. Christiansen (1956) determined this recovery slope by testing at 30 seconds and 15 minutes after exposure.

### C. Other Possible Susceptibility Indices

Most susceptibility tests present each ear with a single specific exposure; either the magnitude of the TTS at a single frequency at some specified time or the slope of the recovery curve over a specified period (or a combination of magnitude and slope) constitutes the susceptibility index.

There are several departures from this paradigm. Rüedi (1954), instead of exposing at only a single level, gave 2-minute exposures to noise at successively higher intensities. Specifically, he exposed the ear for 2 minutes at 80 db SPL, tested the TTS at 1000 and 4000 cps for 2 minutes, exposed for 2 minutes at 85 db SPL, again tested the TTS at 1000 and 4000 cps for 2 minutes, etc., until the TTS reached some criterion value. The intensity that just produced this criterion TTS is called the "critical intensity" by Rüedi, and is taken as the susceptibility index. Such a procedure has two advantages. First, by approaching the criterion TTS gradually, it minimizes the possibility that the susceptibility test may itself produce some permanent loss in the most susceptible ears. Theilgaard (1951) reported that a 30-db permanent loss was apparently caused in an elderly worker by a 5-minute exposure to a 1000-cps tone at 100 db HL (presumably about 115 db SPL). The second advantage is that it precludes the possibility that a large fraction of the test popula-

tion might show no effects whatever from a fixed exposure; obviously, there is no way to judge the relative susceptibilities of two ears both of which show no TTS. Because of these advantages, the stepped-intensity tests seem to be the best method of determining individual differences in TTS; their only drawback is that they take more time than some other test procedures.

Palva (1958) proposed a test in which the exposure was a tone at 30 db SL: 30 db above each listener's threshold. To the extent that such adjustment of the exposure intensity eliminates in part the effect of differences between the conductive part of the aural mechanisms of different listeners, this test might be regarded as determining end-organ susceptibility differences alone. In my opinion, since industrial noises do not obligingly shift their levels to compensate for the worker's initial sensitivity, it would be surprising if such a low-level-exposure test were correlated with susceptibility to PTS from intense noise. But it may turn out to be so, nonetheless; recent studies (Epstein *et al.*, 1962; Katz, 1965) have shown that the short-term TTS produced by low SLs is greater for persons with conductive losses than for those with normal hearing.

A phenomenon quite distinct from TTS is the change in the lateralization of a diotic tone after long monotic stimulation. The literature on this "peristimulatory fatigue," which I shall refer to as "adaptation," has recently been reviewed by Small (1963). First one determines the intensity of the tone in the right ear (RE) that, when paired with a tone of the same frequency in the left ear (LE), will give rise to a percept that seems to be exactly in the middle of the head. The left ear alone is next exposed for several minutes to the tone, and then one quickly determines how much less intense the RE tone must now be in order to once more give rise to the median-plane lateralization; this difference is the measure of adaptation.

Tanner (1955) advocates a test in which both the adaptation and the TTS produced by a 1000-cps tone at 100-db "intensity" (probably HL) are measured. Tanner reports a negative correlation between the two indices: the greater the adaptation, the lower the TTS, and vice versa.

Another measurement that might be expected to be related to susceptibility is the degree of contralateral remote masking (CRM). If an intense high-frequency octave band of noise (e.g., 2400–4800 cps) is put into one ear, it is found that more masking of a low-frequency tone in the opposite ear occurs than can be accounted for by transcranial conduction of the noise (Ward, 1961). No matter whether this occurs either because of contraction of the middle-ear muscles or because of interference, at some station in the auditory pathway, between excitation initiated by the test signal and the massive neural discharge from the

noise-exposed ear, on face validity grounds one can argue that CRM should be correlated with the TTS produced.

#### D. Susceptibility or Susceptibilities?

Up to this point, the discussion has assumed, more or less tacitly, that susceptibility is a unitary trait—that the person most susceptible to damage by low-frequency pure tones will also be most susceptible to damage by a high-frequency tone, a broad-band noise, high- and low-frequency octave-band noises, intermittent tones and noises, firecrackers, rivetting, and so on. Unfortunately, nature is this simple only infrequently, and this is probably not one of the times. In 1951, Greisen administered 5 different pure-tone susceptibility tests to 24 listeners. The tests used by Greisen were those of Peyser (1943), Theilgaard (1951), Wilson (1944), and two of his own (see Table I). These tests involved exposures to tones of 1000, 1500, 2000, and 3000 cps. The only significant correlation (0.78) that emerged from his data was between Greisen's two tests, that is, between the TTSs produced by a 5-minute exposure to 3000 cps at 80 and at 90 db HL. Furthermore, as long ago as 1920, Flügel had amply demonstrated that the two ears of a given observer might display quite different susceptibilities to TTS from the *same* exposure.

It is therefore clear that if susceptibility to permanent loss can be predicted by individual differences in TTS, we should expect to find that several susceptibilities exist—and for each ear, not just each observer. The research to be described below is an attempt to determine the number of indices necessary to specify the susceptibility of a given ear to TTS. Specifically, the broad experimental question is this: To what extent, if any, can a given ear be characterized as “more susceptible” or “less susceptible” *in general* to TTS, and to what extent must one instead speak of susceptibilities specific to a given frequency, intensity, and temporal pattern? In a sense, it is only an elaboration of Greisen's study, but with (1) a larger sample (2) of only ears having normal sensitivity, using (3) more susceptibility tests, (4) better apparatus than the typical clinical audiometer, and (5) the modern statistical techniques of factor analysis to separate “general” from “specific” susceptibilities.

## II. PROCEDURE

### A. Apparatus

Auditory thresholds were determined by means of the modified method of limits (Békésy audiometry) wherein the listener causes the tone to vary between audibility and inaudibility. Specifically, the apparatus consisted of the following, in sequence: (1) audiooscillator; (2)

electronic switch, set to produce 250-msec tone pulses with rise and fall times of 20 msec, separated by 250 msec of silence; (3) motor-driven recording attenuator controlled by the listener; during threshold testing the attenuation was continually increasing or decreasing—depending on whether or not the listener was pressing a response button—at 4 db/sec; (4) amplifier; (5) fixed attenuator; (6) switch box; (7) PDR-8 earphones set in MX-41/AR cushions. Fatiguing stimuli were presented by connecting the earphone directly to the output of an amplifier fed either by another pure-tone oscillator or by a noise generator followed by two sets of passive filters (the skirt characteristic of the resulting noise band was 60 db/octave). Calibration of tones and noises from the earphone was performed routinely by means of a frequency counter and a standard condenser microphone system coupled to the earphone by means of a brass ASA Type 1 6-cc coupler. Although the two earphones were nearly matched in response, slight adjustments were made throughout the course of the studies to insure that the same overall coupler SPL was produced by each phone in a given experiment. Distortion of the electrical signal delivered to the earphone was determined to be just under 0.3% at the highest levels of pure tone employed. Tests before and after the 8 months required for completion of the experiments showed only negligible changes in any system components.

All testing was performed in a double-walled audiometric booth. Ambient noise levels were below those required for measurement of threshold SPLs of 0 db at frequencies of 1000 cps and above. The subject was seated with his back to the window in the booth, so that the experimenter could make sure that the earphones were donned reasonably correctly and that the subject did not readjust the phones once an experiment had begun.

For the experiments involving TTS produced by impulses (Week 18 below), a special high-intensity driver unit coupled to an exponential horn was used. A high-capacity condenser was periodically discharged through a thyatron in series with this speaker. The listener's ear was placed in a constant position just beyond the end of the exponential horn. This apparatus, together with the acoustic waveform produced, are described in greater detail in an earlier report (Ward *et al.*, 1961).

### B. Subjects

The listeners were recruited from the universal source—the sophomore class of the University—by advertising. The hearing of each applicant was tested to determine that it was “normal” (within 15 db of the zero point on standard audiometers). Three applicants were rejected because of hearing threshold levels exceeding 20 db; all others were

accepted. The study began with 27 men and 28 women; three of each sex did not complete the series, so the final sample of listeners consisted of 24 men and 25 women. The low attrition can be ascribed to the use of monetary incentive: the listeners were paid \$1.50/hr monthly, with a bonus of \$0.75/hr paid in a lump sum at the termination of the study. Those who did abandon the project all did so during the first three weeks of testing. The worst exposures were deliberately given early in order to weed out the timid early in the game; however, the TTSs suffered by the quitters were not greater than the average of the rest of the listeners. Nor were their resting thresholds any different. One can therefore assume that there is no essential correlation between sensitivity and the tendency to discontinue the series, and hence that no bias toward low susceptibility existed in the final sample.

### C. Method

Each listener was tested (unless otherwise noted) on each of 23 weeks as follows:

*Week 0: control threshold data.* Detailed audiograms were obtained at quarter-octave intervals from 0.25 to 8 kc and at 10 and 13 kc. Each frequency was tested for at least 20 seconds, more generally for 30 seconds.

*Week 1: 2.8-5.6 kc noise in 1-minute steps, 85-125 db SPL (monaural), plus CRM at 500 cps.* This procedure was patterned after the tests devised by Rüedi (1954). Following preexposure tests of the RE at all frequencies (quarter-octaves) from 2.4 to 13 kc, a 2.8-5.6 kc octave band of noise at 85 db SPL was presented for 1 minute. Then for 1 minute the TTS at 6.7 kc was followed. Next the ear was, without pause, exposed to 1 minute of the same noise but at 95 db SPL, and again the TTS at 6.7 kc was followed for 1 minute, etc., through an exposure SPL of 125 db. After this final exposure and the 1-minute test at 6.7 kc, 20-second samples of shifted threshold at the other frequencies from 4.7 to 13 kc were determined in a fixed sequence. The entire procedure (including the preexposure tests) was completed without removal or adjustment of the earphones. Then, after a short break, the whole procedure was repeated on the LE. (In monaural exposures, the same phone was used for both RE and LE in order to insure that the same noise was presented to all ears.) While the noise was being presented to one ear, the listener was kept busy tracking a 500-cps tone in the other ear in a determination of CRM.

*Week 2: 0.70-1.4 kc noise in 1-minute steps, 95-125 db SPL (monaural), plus contralateral masking at 6.7 kc.* The procedure here was analogous to that in Week 1, except that the noise was centered at 1 kc,

the major test frequency was 2 kc, terminal TTS was determined for frequencies from 1.4 to 4 kc, and the CRM was measured at 6.7 kc (that is, at a frequency higher than the noise).

*Week 3: 1.4-2.8 kc noise in 1-minute steps, 95-125 db SPL (monaural) plus CRM at 500 cps.* Same general procedure as Week 1; major test frequency 4 kc, TTS audiogram 2.8 to 8 kc, CRM at 500 cps.

*Week 4: controls.* This was the half-week just before the Christmas holidays, so less than half the listeners could be tested. Therefore, some control experiments were made. First, the right ears were subjected to the identical procedure of Week 1, primarily in order to determine whether or not any systematic changes as a function of experience were occurring. (They were not, according to the results.) Then, after a rest, the LEs were tested with the procedure of Week 3, but with the test tone continuous throughout instead of interrupted.

*Weeks 5-7: 15 minute 1.4-2.8 kc noise at 106 db SPL (earphone exposure) or 100 db SPL (field exposure) (binaural).* In order to determine the relative test-retest reliability of earphone versus field exposures to noise, the next three sessions involved 15-minute binaural exposures via earphone in one case, or by sitting in front of a loudspeaker in the other. The earphone level was set at 106 db as measured with the ASA Type 1 coupler, while the field was adjusted to be 100 db SPL (using the same microphone but with no coupler) at the listener's ear level. The listener's head was 3 ft directly in front of, on the axis of, and facing, the high-frequency element of a large loudspeaker (Altec Voice of the Theater); constancy of position was assisted by a fixed headrest.

On Week 5, half the listeners were given field exposures and half phone. On Week 6 exactly the same procedure was followed. Then on Week 7 those previously exposed via phone were given field noise, and vice versa. Because two ears were being tested, resting and shifted thresholds were determined only at half-octave intervals from 1.4 to 5.6 kc. In addition to the immediate postexposure threshold shifts, a final shifted audiogram was measured during the period from 13 to 17 minutes following the exposure.

*Week 8: 15 minute 1.4-2.8 kc intermittent noise at 109 db SPL, 50% duty cycle (binaural).* Since individuals differ in the degree to which TTS is lessened when the exposure is intermittent (Ward, 1962a) the same procedure used for earphone exposure on Weeks 5-7 was followed, except that the noise was now on for a second, off for a second, etc. The level was raised 3 db, so that the total exposure energy was constant, although it was known that such a procedure would still not produce TTSs equal to those from 106-db continuous noise.

*Week 9: effect of hair on field exposures.* It had been found in Weeks 5-7 that the men showed more TTS than women in the field situation

but not when exposure was via earphone. In order to evaluate the possibility that the women were protected by their longer hair, the field study of Weeks 5-7 was repeated on most of the listeners, but with *hair length versus sex* reversed: the women wore surgical caps with their ears exposed, while Beatle wigs, providing hair over the ears, were worn by the men. This reversal proved to have no effect on the TTS produced, so the sex-related difference in TTS in this case is apparently due to the fact that men's ears project farther from their heads than do women's (to be discussed later).

*Week 10: 3 minute 1.4-2.8 kc noise at 116 db SPL (binaural).* Testing as in Weeks 5-8, but binaural earphone exposure for 3 minutes at 116 db SPL. In all subsequent tests, all exposures employed earphones.

*Week 11: 3 minute 2.8 kc pure tone at 116 db SPL (binaural).* Exactly as in Week 10, but with the 1.4-2.8 kc noise replaced by a 2.8 kc pure tone (the pattern of TTS produced by an octave band of noise is more nearly like that produced by a tone at the upper cutoff frequency than one at the center frequency of the band (Ward, 1962b)).

*Week 12: 15 minute 0.35-0.70 kc noise at 120 db SPL (binaural).* TTS was measured at half-octave frequencies from 0.5 to 2 kc during the time from 0 to 7 and 13 to 17 minutes post exposure.

*Week 13: 5 minute 0.70 kc pure tone at 125 db SPL (binaural).* Testing as in Week 12.

*Weeks 14-15: 3 minute 1.4 kc pure tone at 115 db SPL (monaural and binaural).* Half the listeners were run binaurally on Week 14 and monaurally on Week 15, the other half vice versa. Pre- and postexposure thresholds from 1 to 4 kc.

*Week 16: dichotic versus diotic exposures.* On this week, half the group was exposed for 3 minutes diotically (0.70-1.4 kc noise at 120 db SPL to both ears) and half dichotically (noise to RE, 120-db 1.4-kc tone—as in Weeks 14-15—to LE). The differences among the TTSs produced by these dichotic and diotic exposures and by the monotic exposures of Week 17 have already been reported (Ward, 1965a).

*Week 17: 3 minute 0.70-1.4 kc noise at 120 db SPL (monaural).* Same test procedure as Weeks 14-15.

*Week 18: impulse noise.* On this week, the listeners were exposed to successively higher intensities of clicks until the TTS at 4 kc 30 seconds after cessation reached 30 db. In a procedure similar to that used in Weeks 1-3, each ear was exposed for 1 minute to clicks whose peak intensity was 136 db SPL at a rate of 1 click per 2.4 seconds (a total, therefore, of 20 clicks). This was followed by 1 minute of testing at 4 kc; if the TTS at 30 seconds was not 30 db or more, then at the end of the 1-minute test period the ear was exposed at a level about 3.3 db higher.

This was continued either until the TTS at 30 seconds reached 30 db or the maximum level (156 db peak) had been reached. A terminal audiogram was taken, but there is little one can do with these data because of the wide range of exposures.

*Week 19: low-level TTS (after Palva, 1958).* The short-term TTS at a given frequency produced by a 1-minute exposure to a 40-db SL tone of the same frequency was measured for frequencies of 8, 4, 2, 1, and 0.5 kc, in that order. The procedure for each frequency was as follows: (1) measure threshold for interrupted test tone; (2) determine threshold for continuous test tone; (3) expose for 1 minute at 40 db SL (by simply changing the master attenuator by 40 db); (4) follow for 1 minute the TTS with continuous test tone; and then (5) determine the TTS for an interrupted test tone (from 1 to 1.5 minutes).

*Week 20: adaptation and TTS from 5 minutes of 1-kc tone at 110 db SPL (after Tanner, 1955).* In addition to the TTS produced at frequencies half an octave apart (from 1 to 5.6 kc) by a 5-minute exposure to a 1-kc tone at 110 db SPL, measurements of adaptation were made from 0.5 to 1, 2.5 to 3, and 4 to 4.7 minutes after beginning of the adapting exposure. This was done by turning on an interrupted 1-kc tone in the control ear during those times. The specific instructions were these: "In this test, you are going to have to try to adjust the loudness of a beeping tone to be the same loudness as a continuous tone in the other ear. After the usual preexposure tests, you will hear a steady tone in the RE, which will be on for 5 minutes. From time to time, I will turn on the beeping tone in the other ear, and the beeping tone will get louder and louder until you press the button. I want you to press the button just as soon as the beeping tone seems just barely louder than the steady tone, and hold the button down until the beeping tone seems softer, and so on. In other words, instead of making the beeping tone become alternately audible and inaudible, make it go back and forth across 'equally loud.' Both before and after the steady tone, of course, you will use the button to trace your threshold as usual."

*Week 21: composite noise (binaural).* By this time even casual inspection of the data that had been accumulating showed clearly that susceptibility was not a unitary trait. Those listeners showing the most TTS from one octave band of noise were not always those showing the most effect from another. The question that arises, then, is this: Must one determine susceptibility to each octave band of noise in separate experiments, or will a single exposure to broad-band noise give essentially the same results? That is, if octave band  $B_1$  produces the most effect at frequency  $f_1$  on listener X, band  $B_2$  the most effect at  $f_2$  on listener Y, and band  $B_3$  the most effect at  $f_3$  on listener Z, will an exposure to  $B_1 + B_2 +$



$B_3$  also produce the greatest relative shift at  $f_1$  in X, at  $f_2$  in Y, and at  $f_3$  in Z? Accordingly, a composite noise was developed by adding (1) the outputs of two channels of a tape recorder on which were prerecorded noises of 1.4-2.8 kc and 2.8-5.6 kc, respectively, to (2) 0.70-1.4 kc noise directly from the noise generator and filters. The octave-band levels of the final output were 125, 117, and 111 db SPL for the 0.70-1.4, 1.4-2.8, and 2.8-5.6 kc bands, respectively. TTS was measured at half-octave intervals from 1 to 8 kc.

*Week 22: control—repetition of Week 1 (RE), and composite noise, monaural (LE).* As a final check on learning processes or "toughening" of ears as a function of the weeks of exposure, the right ears of all listeners were run on the schedule of Week 1. And since the TTS produced by binaural exposure to the composite noise (Week 21) had been substantially below what one might have predicted from previous data, the LEs were exposed to the composite noise, in order to assess the monaural-binaural difference in TTS from the composite noise.

#### D. Comments on Procedure

Crucial to any study of individual differences such as this one is the necessity of insuring that conditions remain as constant as possible. Thus, for example, it would be desirable if the earphones could be placed on each listener in exactly the same manner each week. Some preliminary effort was devoted to testing the notion that constancy might be insured by using a probe tube and microphone permanently set in the earphone cushion in order to measure and hold invariant the sound field under the earphone. However, this procedure proved so difficult that it was abandoned. Instead, the listeners were instructed at length about the necessity for a constant position of the earphones and were required during Week 0 to practice removing and replacing them, toward the objective of a "most comfortable fit."

Another possible confounding variable arises from the fact that each listener was tested at the same hour of the same day each week. If there happened to be a diurnal variability in susceptibility to TTS that was common to all the listeners, then the morning subjects, for example, might show more effect than the afternoon listeners. A preliminary study (Ward, 1964) showed that no diurnal variability in threshold sensitivity exists, so it is not likely that susceptibility would vary cyclically.

### III. TREATMENT OF DATA

With 35 to 60 datum points gathered for each ear every week, somewhat more than 100,000 were available at the end of the experiment. In order to be amenable to factor analysis even by a computer, this number

obviously had to be reduced by at least a factor of 10. Accordingly, some preliminary correlational analyses of the data were performed.

#### A. The Threshold

If one is to use a shift in threshold as the main variable in analysis, the first question to be considered is this: Should TTSs be calculated on the basis of the preexposure threshold (PE) measured on the day concerned, or in relation to the *average* PE? Both alternatives have certain advantages. If the phones are not moved during the entire run, it can be argued that the effect of slight differences in phone placement is eliminated by using the that-session value of PE. On the other hand, to the extent that day-to-day variability of threshold is not correlated with earphone placement, but is due either to slight amounts of TTS caused by the noises of everyday life (such as riding in a noisy car just before coming in for test), or to internal factors not now measurable and so called "chance," then basing all TTSs on the average PE measured over the 23-week period might be the better course.

One answer to this question can be sought in the test-retest control runs. The standard deviations of the differences between Weeks 5 and 6 and Weeks 1 and 22 were calculated for both the TTS (relative to that day's PE) and the shifted threshold (which is the same as using an average preexposure). Table II shows the results for Weeks 5 and 6.

TABLE II

*Unbiased Estimates of Standard Deviations of Differences in Threshold Shifts and Shifted Thresholds between Weeks 5 and 6 (Test-Retest). All Entries in Decibels.*

Test frequency	Phone exposure			Field exposure		
	Threshold shift	Shifted threshold	Shifted threshold	Threshold shift	Threshold shift	Shifted threshold
2	4.1	2.7	2.7	4.35	4.35	3.35
2.8	4.3	2.55	2.55	4.25	4.25	3.35
4	3.9	4.5	4.5	5.1	5.1	3.6
5.6	4.55	6.85	6.85	7.0	7.0	5.15

When the experiment involved earphone exposure (columns 2 and 3), shifted threshold was more reliable for test frequencies of 2 and 2.8 kc, but threshold shift appeared more stable at 5.6 kc. Under field exposure conditions, shifted threshold was less variable than threshold shift at all frequencies. However, in this case the phones were necessarily removed and replaced between the pre- and postexposure testing, so this would tend to add to the variability of the threshold shift measures. The re-



sults of the comparison of Weeks 1 and 22, 6 months apart, agreed with the high-frequency data in Table II: the TTS showed greater stability than shifted threshold, although for both indices the variability was higher than when the test and retest were performed on successive weeks ( $\sigma = 7$  db for TTS and 9 db for shifted threshold at frequencies from 4.7 to 8 kc).

The data therefore do not provide an unambiguous answer to the question. Taken at face value, these results imply that for maximum reliability one should calculate high-frequency TTSs (above 4 kc) relative to a PE measured at the same sitting, but low-frequency TTSs relative to an average PE measured over several sessions. In view of the fact that in applying a susceptibility test to a large number of industrial workers, one would in general have available only the that-day PE, it was decided to analyze the results in terms of TTS based on the threshold of the day concerned.

#### B. One Frequency or Many?

One way to reduce the magnitude of intraobserver chance variability is by calculating the average TTS over several frequencies instead of using a single frequency. For example, the test-retest correlation between Weeks 1 and 22 was only 0.66 for the TTS at 5.6 kc 1.5 minutes after exposure, but was 0.77 for the average of the TTSs at 5.6, 6.7, and 8 kc. Accordingly, average TTSs at two or three frequencies showing the most shift (the most for the group, of course, not the individual) were calculated for each ear on each test. The correlations between this average TTS and the TTS at the single frequency of maximum shift was calculated for each session; the values of  $r$  ranged from 0.78 to 0.95 with a median of 0.86.

#### C. What Value of Recovery Time?

Thresholds at the frequencies of maximum shift were always measured at least twice—during the period between 1 and 2.5 minutes and between 3.5 and 6 minutes after exposure. In addition, the TTS about 15 minutes after exposure was measured when time permitted, that is, on weeks involving binaural exposures. Test-retest comparisons revealed no difference between the reliability of either maximum or average TTS whether measured at 2 or at 5 minutes of recovery: the correlation was 0.77 between Weeks 1 and 22 in either case for the average TTS, 0.64 for the maximum TTS (at 5.6 kc). Correlations between TTSs at about 2 and about 5 minutes for specific tests ranged from 0.73 (Week 12) to 0.96 (Week 13), with a median of 0.90. Removing and replacing the phones, of course, tended to lower the correlation coefficients: the me-

dian  $r$  between TTSs at 2 and at 15 minutes was 0.85. Analysis showed no significant relation between the size of these correlation coefficients and the magnitude of the TTS, the frequency region involved, whether the exposure was to tone or to noise, or whether the exposure was shorter (3 minutes) or longer (15 minutes). The only consistent relation observed was that the men showed higher correlation coefficients than women (in 18 out of 22 comparisons).

Since the evidence indicates that TTS at either about 2 or about 5 minutes will prove as reliable as the other, the TTS at about 2 minutes was used. (The third alternative, averaging the two, was considered, but appeared to involve more clerical work than it would be worth.) In the following discussion, the term TTS<sub>2</sub> will be used to indicate the average TTS at the 2 or 3 frequencies most affected, measured 1–3 minutes after exposure.

#### D. One Ear or Two?

Since the two ears of a given observer are not completely independent, we do not have results from a sample of 98 ears to analyze, but only from two groups of 49 ears. All comparisons reported to this point represent the average between the RE analysis and the LE analysis, calculated separately.

Correlation coefficients between RE and LE were determined for TTS at the single frequency of maximum shift: these were typically between 0.6 and 0.7, with a median of 0.63. The highest correlation was 0.78 (Week 13—the TTS at 1.2 kc 2 minutes after exposure to a 700-cps tone); the lowest 0.21 (Week 11—TTS at 4 kc 2 minutes after 2800-cps tone). Perhaps the two ears of a given observer could be assumed to be independent in regard to the TTS produced by a 2800-cps pure tone, but for most other stimuli used, from 40% to 50% of the variance among the LEs of the listeners can be accounted for in terms of the TTS produced in the REs.

#### E. Summary of TTS Measures

The main TTS data have at this point been trimmed and compressed to 1568 items: 16 average TTSs for each of the two ears of 49 observers. Of the 22 weeks of experimentation, Weeks 4, 7, 9, 16, and 22 involved control runs in which all ears did not receive the same exposure. Furthermore, the measurements of TTS from impulse noise (Week 18) involved unequal exposures, since exposure was terminated as soon as a criterion TTS was reached. Week 19 was concerned with TTS from low intensities, and so will be considered later. On the other hand, the Week 21 exposure, to broad-band noise, produced TTS at both middle and high fre-

quencies, so two separate indices from this week are included in the matrix: average TTS<sub>2</sub> at 2.8, 4, and 5.6 kc, and average TTS<sub>1</sub> at 1, 1.4, and 2 kc.

### F. Critical Intensity

The stepped-intensity tests (Weeks 1, 2, 3, and 18) all involved 1-minute exposures alternating with 1 minute of testing. As an arbitrary critical intensity (CI), I used the intensity that produced a TTS of 10 db 30 seconds after cessation of exposure. The criterion test frequency was 6.7 kc for Week 1; 4 kc for Weeks 3 and 18. Thus if a given ear showed a TTS of 8 db after a 1-minute exposure at 105 db SPL, and a 13-db TTS after 115 db SPL, the CI was found by interpolation to be 109 db SPL. Unfortunately, on Week 2, only 63 of the 98 ears reached this criterion TTS at all. Therefore a CI for this week (0.70–1.4 kc exposure) was not included in the final matrix of data. The problem of failure to reach criterion was not encountered on Weeks 1 and 3, but again posed a problem on Week 18 (click exposure). Individual differences in susceptibility to TTS from impulses are typically huge (Reid, 1946; Fletcher and Riopelle, 1960; Ward *et al.*, 1961), and this group of subjects proved to be no exception. Although the most sensitive ear showed a TTS at 4 kc of 15 db after 20 clicks at 143 db peak SPL, 14 ears failed to show even a 5-db shift after 20 clicks at 156 db SPL. The CI to clicks for these 14 was arbitrarily called "160" for purposes of analysis. Such an artificial truncation of the distribution of CIs is unfortunate, but since only about 15% of the sample was involved, it should not introduce an intolerable degree of error in the analysis.

### G. Low-Level TTS

Week 19 involved the TTS produced at the exposure frequency after 1-minute exposures at 40 db SL to octave frequencies between 8 and 0.5 kc. Analysis showed that the mean results at 8, 4, and 2 kc were indistinguishable, although 1 and 0.5 kc gave slightly less TTS. Therefore the index used for this week was the TTS 30 seconds after cessation of exposure, averaged over 8, 4, and 2 kc. The correlation between RE and LE indices was 0.65, a value of approximately the same magnitude as the RE-LE correlation for TTS from high intensities.

### H. Adaptation

On Week 20, the amount of adaptation produced by 1 kc at 110 db SPL was measured 0.7, 2.7, and 4.4 minutes after onset of the exposure tone. Two indices were generated from this data, the magnitude of the adaptation at 2.7 minutes, and the change in adaptation from 0.7 to 4.4

minutes. The rate of adaptation, rather than its absolute value, might conceivably be a good index of susceptibility.

### I. Recovery Slope

By the same token, the rate of recovery from TTS might be an important facet of susceptibility. Given two individuals with the same ini-

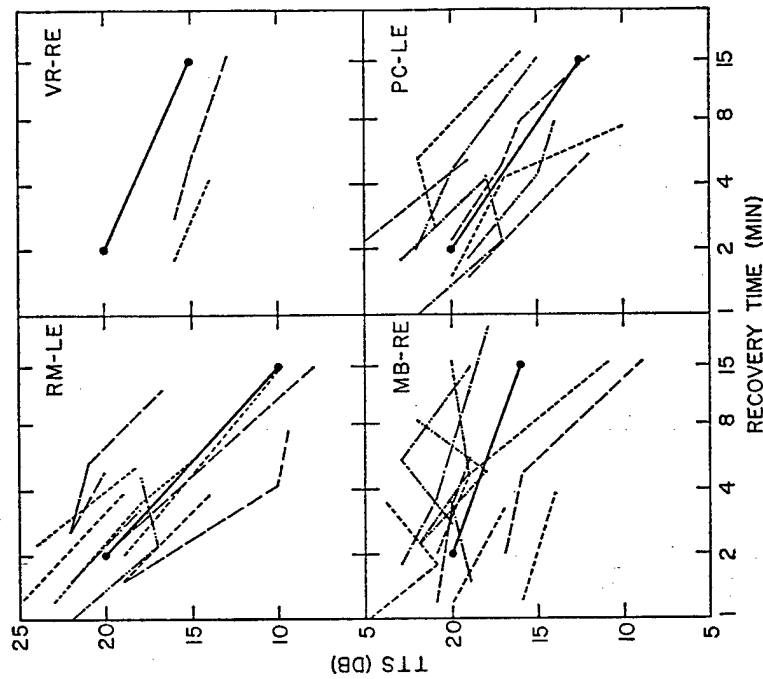


FIG. 1. Illustration of process of deriving individual average recovery slopes. The fine lines represent actual data for a single given frequency, the heavy solid lines the "typical" curve drawn by eye as the experimenter's best estimate of what the recovery slope would have been, beginning with a TTS of exactly 20 db at 2 minutes.

tial TTS, it seems reasonable that the one showing the more rapid recovery should be more resistant to permanent damage. As one would expect, the actual data were unable to provide recovery curves beginning at a specific TTS<sub>2</sub> for all ears; therefore an average recovery slope for an initial TTS<sub>2</sub> of 20 db was developed for each ear by the following procedure. On a single sheet, all data showing the course of recovery of a particular ear from TTS<sub>2</sub> of anything near 20 db were plotted (TTS

versus the logarithm of recovery time). Then a line was drawn by eye, from a TTS of 20 db at exactly 2 minutes, to that at 15 minutes. Although this process undoubtedly introduces some experimental error, the alternative—an even more tedious arithmetic instead of graphic solution—was out of the question.

Figure 1 illustrates several examples of this graphic determination of recovery rate. The top left panel shows one of the easiest sets of data to draw a line through: the actual data are all quite consistent, and bracket the desired TTS<sub>2</sub> of 20 db. The top right panel shows one of the worst; only twice was this ear given as much as 15 db of TTS<sub>2</sub> during the entire experiment, so the estimated function in this case has to be based only on those two curves. The lower left panel is difficult to average because the data are so erratic; however, this ear clearly shows less recovery than most during the first 15 minutes. The final example (lower right) represents about the typical amount of ambiguity in the data for a single ear.

The TTS remaining at 15 minutes was taken as the index; therefore, a high value represents a slow recovery.

#### J. Contralateral Remote Masking

On Weeks 1 and 3, respectively, the contralateral masking of a 500-cps tone produced by 2.8–5.6 and 1.4–2.8 kc noise was measured. A CRM index for each ear was derived by averaging the CRM at 30 seconds (i.e., halfway through the 1-minute exposure) produced by the 125-db exposures in the other ear. Although the correlation between the CRM for Weeks 1 and 3 was only 0.70 for the REs and 0.58 for the LEs, taking the average CRM can be supported by considerations of expediency—if CRM is to be of any value in predicting susceptibility, it must be expressible as a single index.

#### K. "Distortion"

During Week 2, the contralateral threshold shift at 5.6 kc concomitant with the 0.7–1.4 kc band of noise was determined. This masking of a high-frequency tone by a contralateral low-frequency noise presumably depends in part on the amount of harmonic distortion produced within the auditory system as well as on central masking. Therefore the shift 30 seconds after onset of the 125-db noise step was entered as a "distortion" index.

#### L. Preexposure Thresholds

In extreme cases, it is clear that an ear with an elevated threshold will show less TTS than a normal ear. When the loss is due to conductive

factors such as middle-ear blockage, then of course the effective level of the sound reaching the cochlea will be reduced. On the other hand, when the loss represents a sensorineural deficit, then there is less shift possible. Therefore the battery of indices for factor analysis included average thresholds corresponding to frequencies used in measuring TTS: average thresholds at (a) 0.7 kc, (b) 1 and 1.4 kc, (c) 1.7, 2, and 2.4 kc, and (d) 2.8, 4, and 5.6 kc. The values were derived from all measurements of threshold throughout the entire series.

#### M. Békésy Excursion

Individuals differ greatly in their habitual response patterns in the Békésy threshold testing situation. The extremes may be characterized as the cautious and the gamblers. The cautious wait until the test tone is clearly audible—perhaps waiting until they hear three consecutive tone pulses—before pressing the voting button. Similarly, they make very sure that the tone has indeed disappeared before releasing it. The gambler, by contrast, presses the button as soon as he hears *anything* resembling what he is listening for, and may release it when he merely *thinks* that the next pulse will be inaudible. The use of an interrupted test tone tends to limit the gamblers somewhat, since the listeners must attend to a rhythm as well as the tone per se, but it places no restriction on the cautious. In the present case, the lowest average peak-to-valley excursion found was 4 db, which, since the attenuator was moving at 4 db/sec and there were two pulses per second, indicates that even the most inveterate gambler has to hear two consecutive pulses in order to keep the rhythm in mind (he presses after hearing one pulse, but then also hears the next one). Two of the girls consistently ranged over 11 db, which means that they were probably waiting until they had heard five pulses before pressing the button.

The peak-to-valley excursions for preexposure thresholds at 4 kc were measured for both REs and LEs in Weeks 1, 9, and 22, in order to sample the variability over the series. All 6 of these measurements were averaged to provide a single index for each listener, rather than for each ear. This combination of RE and LE results assumes that excursion exponent is dependent on critical factors as described above rather than on such ephemeral constructs as "recruitment at threshold."

#### N. Sex and Ear Projection

Inspection of the data showed that the average results of men and women often differed markedly. These sex-linked differences will be discussed in detail in another article. However, for purposes of factor

analysis, one of the variables included was sex: males were designated 1; females 2.

The final variable incorporated into the battery was the distance that the ears projected from the head: the perpendicular distance from the skull to the rear edge of the auricle, on a line even with the external canal. Although pinna projection, as indicative of the ear's tendency to amplify sound in the same manner as an ear trumpet, might be expected to apply only to the field exposures of Weeks 5-7, it is not unlikely that the correlation might be high between the dimensions of the external ear and those of the middle ear. If so, this index might prove to be related to susceptibility to TTS even when the pinna itself is flattened against the head by the earphone.

#### IV. ANALYSIS

In all, then, 32 indices were calculated for each of the 49 REs and 49 LEs. These particular indices were entered on IBM cards, and an analysis was made using the computer facilities of the Numerical Analysis Center of the University of Minnesota. By means of the UMSTAT-55 program, the following sets of statistics were derived for both the RE and LE data:

- (1) means,
- (2) standard deviations,
- (3) correlation matrix,
- (4) principal-factor analysis,
- (5) varimax-factor analysis.

The first three of these sets are conventional enough to require no comment. The two types of factor analysis, however, are perhaps less well known. Briefly, the principal-factor solution seeks out a single general underlying factor on which as many as possible of the 32 individual indices—in 32-dimensional space—show significant loadings. The varimax solution, on the other hand, involves mathematical rotation of the correlation matrix in many dimensions in an attempt to ferret out a number of orthogonal (independent) factors that will account for more of the variance than is possible with a single factor (for a discussion of factor analysis, see Harman, 1960). Ideally, the principal-factor analysis should indicate to what extent the interindividual variability of all the 32 indices can be explained by a fundamental difference in *general* susceptibility, while the varimax will suggest how many of our 32 indices can be discarded because they are redundant—i.e., because they measure the same aspect of susceptibility.

#### A. Distributional Statistics

Table III gives the mean, standard deviation, and range, for each of the 32 variables, as well as a succinct description of each. The number at the extreme left in bold-face type is an arbitrary one that will be used in what follows to denote the variable in question. Note that the TTS<sub>2</sub> indices are listed first, and in order of increasing exposure frequency rather than in temporal order; this was done in order to facilitate later an intuitive understanding of the relation between the correlation matrix and the factor analyses.

It was hoped that each major TTS test would produce an average of between 10 and 20 db of TTS in order to minimize both (a) the number of ears showing zero shift (and hence an indeterminate relative susceptibility), and (b) the probability that some ears would suffer so severe a TTS that a slight permanent loss might result. In general, the data of Table III show that this objective was fairly well attained. In particular, no permanent effects were produced; the most severe TTS<sub>2</sub> produced (51 db, 15) had completely recovered by the following week. Further evidence that no cumulative effects were produced by these weekly exposures can be inferred from a comparison of the preexposure audiograms for Weeks 1 and 22. The differences between thresholds for these two weeks at all 11 test frequencies from 2 to 13 kc were calculated. Of the 1078 data (98 ears times 11 frequencies), 74 exceeded  $\pm 10$  db. However, 32 of the 74 were apparent *increases* in sensitivity of 10 db or more, so it is unlikely that any of the other 42 shifts—implying a loss of threshold sensitivity—represent anything more than the usual variability at high frequencies ascribable to earphone placement. There were no differences as large as 10 db in either direction for test frequencies 2, 2.4, 2.8, and 3.3 kc.

The lowest mean TTS index is 6, which is the TTS at low frequencies following exposure to the composite noise of Week 21. The low value of TTS can be ascribed partly to the relatively low TTS-producing power of low-frequency noise, but it is also due to the use of binaural instead of monaural exposure and to the fact that TTS at these frequencies was not measured until 4 minutes after exposure. With 25 of the 98 ears showing an average TTS of 2 db or less, the distribution is clearly truncated.

Results from the right and left ears are quite similar. The only difference worth mentioning is that the average LE TTSs for all the 8 tests involving exposure frequencies at or below 1.4 kc are larger than those for the REs ( $p = 2^{-8}$ ). This is true for binaural exposures as well as monaural exposures and so cannot be attributed to the fact that in

TABLE III  
Distributional Statistics for the 32 Variables Subjected to Factor Analysis

Variable	Description <sup>a</sup>	Week		Right ear		Left ear		Range	
		Mean	S. D.	Mean	S. D.	Mean	S. D.	Min	Max
1	TTS <sub>1</sub> (1, 1.4); 15' 120 db: 0.35-0.70 noise	12	8.3	4.8	10.6	5.9	10.6	-3	23
2	TTS <sub>1</sub> (1.4, 2); 5' 125 db: 0.70 tone	13	14.9	8.4	16.0	9.1	16.0	1	43
3	TTS <sub>1</sub> (1.4, 2); 5' 110 db: 1.0 T mon.	20	13.6	5.4	13.9	6.2	13.9	2	28
4	TTS <sub>1</sub> (1.7, 2, 2.4); 1' steps: 0.70-1.4 N mon.	2	6.7	3.1	7.8	3.3	7.8	-1	16
5	TTS <sub>1</sub> (1.7, 2, 2.4); 3' 120 db: 0.70-1.4 N mon.	17	10.4	6.0	10.7	5.5	10.7	0	29
6	TTS <sub>1</sub> (1, 1.4, 2); 3' 125 db: broad-band N	21	4.3	3.1	4.4	3.3	4.4	-1	15
7	TTS <sub>2</sub> (2, 2.8); 3' 115 db: 1.4 T	14	12.3	6.9	13.4	6.5	13.4	0	31
8	TTS <sub>2</sub> (2, 2.8); 3' 115 db: 1.4 T mon.	15	16.4	7.2	16.7	7.0	16.7	-3	38
9	TTS <sub>2</sub> (3.3, 4, 4.7); 1' steps: 1.4-2.8 N mon.	3	15.6	6.3	16.1	6.4	16.1	-1	37
10	TTS <sub>2</sub> (2.8, 4, 5.6); 3' 125 db: broad-band N	21	8.3	4.1	10.5	4.5	10.5	1	24
11	TTS <sub>2</sub> (2.8, 4, 5.6); 3' 116 db: 1.4-2.8 N	10	12.8	5.3	12.7	5.4	12.7	2	27
12	TTS <sub>2</sub> (2.8, 4, 5.6); 15' 100-db field: 1.4-2.8 N	5-7	18.8	5.5	18.8	5.8	18.8	-1	38
13	TTS <sub>2</sub> (2.8, 4, 5.6); 15' 106-db phone: 1.4-2.8 N	5-7	21.1	4.9	19.7	4.2	19.7	10	34
14	TTS <sub>2</sub> (2.8, 4, 5.6); 15' 109 db R = 0.5: 1.4-2.8 N	8	10.2	4.4	9.2	5.7	9.2	-4	25

15	TTS <sub>2</sub> (4, 5.6): 3' 116 db: 2.8 T	11	19.5	6.9	21.2	7.5	21.2	7	51
16	TTS <sub>1</sub> (5.6, 6.7, 8); 1' steps: 2.8-5.6 N mon.	1	26.6	9.5	25.9	7.8	25.9	4	47
17	Critical intensity for impulse noise (SPL) mon.	18	154	3.9	153	4.6	153	142	160
18	Low-level TTS <sub>2</sub> mon.	19	10.1	2.6	10.3	2.8	10.3	5	16
19	Adaptation (peristimulatory fatigue) mon.	20	47.5	8.3	50.0	9.0	50.0	30	66
20	Change in adaptation	20	2.7	6.5	5.6	6.0	6.0	-9	22
21	Sex (males = 1, females = 2)								
22	Ear projection (mm) (L/R only)								
23	CRM at 500 cps (125-db noise)	1, 3	21.0	4.1	20.0	4.5	20.0	11	31
24	CRM at 6.7 kc (125-db noise)	2	8.1	3.3	7.9	3.4	7.9	-3	16
25	TTS <sub>1</sub> , given TTS <sub>2</sub> = 20 db		10.2	2.1	9.1	1.9	9.1	7	16
26	Békésy excursion extent		7.7	1.4	—	—	—	4	12
27	Preexposure: 0.70 (SPL)		8.2	3.9	9.0	4.7	9.0	0	22
28	Preexposure: 1, 1.4 (SPL)		5.1	3.5	4.5	5.0	4.5	-2	16
29	Preexposure: 1.7, 2, 2.4 (SPL)		6.6	4.0	9.1	4.7	9.1	-2	23
30	Preexposure: 2.8, 4, 5.6 (SPL)		7.5	4.8	9.7	5.5	9.7	-3	26
31	Critical Intensity: 1' steps 2.8-5.6 N (SPL)	1	102.5	6.4	104.2	5.8	104.2	85	125
32	Critical Intensity: 1' steps 1.4-2.8 N (SPL)	3	99.1	6.7	99.7	6.0	99.7	87	123

<sup>a</sup> TTS<sub>2</sub> = temporary threshold shift 2 minutes after cessation of exposure. Exposures binatural unless otherwise indicated. All levels given in decibels SPL. CI = critical intensity; CRM = contralateral remote masking; T = tone; N = noise; mon. = monaural.

monaural exposures the RE was tested first. The LEs have higher thresholds to begin with (29), so we must conclude that LEs are slightly more susceptible to TTS than REs.

### B. Correlation Matrix

Raw intercorrelation coefficients among the 32 variables are shown in Table IV. Coefficients below the diagonal are for the RE data, those above apply to the LE. Only coefficients statistically significantly different from zero are entered; for  $N = 49$ , the 5% confidence limits for significant correlations are  $\pm 0.30$ . Entries in italics indicate that significant correlation coefficients between the two variables concerned were obtained for both sets of data.

The outcome of the factor analyses is, of course, inherent in the data of Table IV, and the reader can see obvious groupings without further treatment. However, it is probably easier to discuss the correlations in the context of the two types of factor analysis.

### C. Principal-Factor Analysis

When subjected to factor analysis, the data yielded the following number of significant factors (eigenvalues greater than unity): males, RE or LE, 10; females RE 9, LE 11; combined sexes, RE 9 and LE 10. However, in the principal-factor analysis, only the first factor is of primary interest. In all 6 analyses, this factor "explained" about twice as much of the total variance as the second most important. Table V presents the loadings of each variable on this principal factor for each of the analyses. Only loadings greater than  $\pm 0.30$  are listed.

The variables showing a significant loading on the principal factor for both the RE and LE analyses include the TTSs produced by all noises and tones used except the highest in frequency (2.8-5.6 kc noise and 2.8-kc tone), sex, ear projection, average threshold at 1 and 1.4 kc, and the critical intensity for Week 3. These variables are designated in Table IV by asterisks.

Clearly, there is indeed a general susceptibility that is characteristic of any given ear, and this general factor is reflected in the TTS produced by exposure to tones or noises of 2 kc or below. The highest average loading is achieved by 7, the average  $TTS_{2.2}$  at 2 and 2.8 kc after a 3-minute binaural exposure to a 1.4-kc tone at 115 db SPL. So if one must select from the literature a single test whose results will be most representative of other tests involving low-frequency exposures, the best choice would appear from Table I to be that of Theilgaard according to Greisen: 1.5 kc at 100 db HL (which in 1951 was just about 115 db SPL) for 5 minutes. It is ironic that Theilgaard himself, of all those in-

TABLE IV  
Intercorrelation Matrix among 32 Indices: RE Data below Diagonal; LE Data above Diagonal.  
Statistically Significant ( $p = 0.05$ ) Coefficients Only

Variable number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
1																																
2	0.61																															
3	0.58	0.53																														
4	0.58	0.53	0.58																													
5	0.58	0.53	0.58	0.58																												
6	0.58	0.53	0.58	0.58	0.58																											
7	0.58	0.53	0.58	0.58	0.58	0.58																										
8	0.58	0.53	0.58	0.58	0.58	0.58	0.58																									
9	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58																								
10	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58																							
11	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																						
12	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																					
13	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																				
14	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																			
15	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																		
16	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																	
17	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58																
18	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58															
19	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58														
20	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58													
21	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58												
22	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58											
23	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58										
24	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58									
25	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58								
26	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58							
27	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58						
28	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58					
29	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58				
30	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58			
31	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58		
32	0.58	0.53	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	0.58	

\* Italicized entries indicate correlation coefficients that were significant for both RE and LE analyses. Decimal points are omitted. Asterisks denote variables showing a significant loading on the first factor of the principal-factor analysis summarized in Table III.

\* Columns 17, 18, and 19 have been omitted because they contained no significant correlation coefficients.

TABLE V  
Loading Exceeding  $\pm 0.30$  for Each of 32 Variables on the Most Salient Factor  
Emerging from Principal-Factor Analysis. Six Separate Analyses.  
Decimal Points Omitted

Variable no.	RE, males	RE, females	RE, all	LE, females	LE, males
1		+63	+43	+58	+44
2	+34	+75	+61	+77	+76
3	+84	+77	+75	+69	+71
4	+37	+70	+59	+69	+73
5	+69	+71	+67	+82	+86
6	+53	+78	+63	+70	+69
7	+89	+69	+78	+78	+81
8	+85	+64	+79	+66	+66
9		+62	+38	+47	+30
10	+37	+71	+58	+62	+60
11	+38	+73	+57	+45	+32
12	+73	+72	+75	+53	+48
13	+44	+53	+51	+46	+42
14	+55	+70	+63	+64	+71
15		+34			
16	-43		-33		
17	-54				
18					
19					
20		-42	-33	-33	
21		+44	+43	+38	
22		-37		-41	-55
23				-33	-50
24	-42			+44	+69
25	+38				
26					
27	-38			-34	-39
28	-49	-57	-50	-46	-53
29	-46	-61	-51	-53	
30		-39		-33	
31	+45	-57			-46
32		-75	-64	-51	-43

investigators proposing susceptibility tests before 1950, is the only one who was not fairly confident that there was a single general susceptibility and that his test measured it best: "It would . . . be quite illusory to characterize the degree of 'auditory fatigue' of a single individual by referring to the magnitude of the effect recorded at a single frequency" (Theilgaard, 1949).

With regard to the four other variables highly related to this general factor, we can probably regard the high loading of 32 as trivial. Since 32

represents the CI for 1.4-2.8 kc noise, and since TTS in any ear increases with intensity of exposure, the high negative loading merely reflects the fact that those ears that reach a criterion TTS at the lower levels will display a greater TTS at any intensity above this.

Variable 28 also shows a negative loading on the principal factor. This is consonant with the correlation matrix of Table IV, which shows that this variable (the average resting threshold SPL at 1 and 1.4 kc) is negatively correlated with 3, 7, and 8; the more sensitive ears tend to show more TTS than those with higher thresholds. Such an outcome would be inevitable if the range of resting thresholds were large: if the differences were due to conductive factors, then those with elevated thresholds would be getting less exposure (at the cochlea), while if the differences were a result of sensorineural loss, there would of course be less shift possible. The fact that a significant loading was obtained here, in the group of ears that were all well within the limits of what we usually call "normal," proves that the measured differences in resting threshold, whether conductive or sensorineural, are at least real, and not just a matter of differences in individuals' criteria for detectability.

The negative loading of 21 (sex) shows, since males were designated 1 and females 2, that females in general have a slightly lower general susceptibility than males. It is interesting that this relation emerged from the total analysis despite the fact that, as shown in Table IV, there was a significant raw negative correlation in both RE and LE data between sex and TTS only for the field exposure (12).

The positive loading of 22, ear projection, is, for some reason, due only to the female half of the sample. In both the RE and LE male analyses, the loading of ear projection was essentially zero. It appears, then, that if a particular woman has large or protruding ears—nearly as big as a man's—she will be more like a man in susceptibility; on the other hand, it apparently does not help a man to have small ears.

In this particular factor analysis, the principal factor was able to explain only 33% of the total communality (7.2/22.0) so it is apparently not an exceedingly powerful common factor. More variance is of course accounted for by a combination of factors as in the varimax solution.

#### D. Varimax-Factor Analysis

Table VI presents the varimax results. To get the data for this table, factors emerging from the RE and LE analyses were inspected and matched as well as possible. (It is clear that in some cases this match was considerably less than perfect, particularly since the RE analysis developed nine factors, while only eight appeared from the LE data.) Then the loadings for corresponding factors in the RE and LE analyses were



TABLE VI  
Significant Loadings of All 32 Variables on Each of 8 Factors Derived by  
Varimax Analysis (Average of Values from RE and LE Analyses)<sup>a</sup>

Variable	Factor							
	$F_a$	$F_b$	$F_c$	$F_d$	$F_e$	$F_f$	$F_g$	$F_h$
1	60			36				
2	65							
3	80							
4	73							
5	86							
6	73							
7	77							
8	72							
9		50						
10	32	62						
11		64						
12		42						
13		55					55	
14		64					36	
15								32
16			70	-31				
17			34					
18								64
19						48		
20						-64		
21				-78				
22				79				
23								
24							-41	
25			-31					
26					-36			
27					65			
28					74			
29					67			
30					49			
31								
32								
Percent of variance	24	16	10	10	11	8	10	7

<sup>a</sup> Decimal points are omitted.

averaged, and all mean loadings exceeding 0.30 were entered in Table VI. The bottom line shows the percent of the total variance—that is, the total variance explainable by 8 factors—associated with each factor. These eight factors will be considered in turn.

$F_a$ : low-frequency TTS. This factor is composed mainly of all TTSs

from exposure to frequencies up to and including 1.4 kc. As in the principal-factor analysis, the threshold sensitivity at 1 and 1.4 kc is also involved. The significant loading on variable 10 probably results from the fact that 6 and 10 are not as independent as most of the other TTS indices, being produced simultaneously by the broad-band composite noise.

$F_b$ : mid-frequency TTS. The variables involved in this factor are the TTSs (9-14, inclusive) and the CI (32) from 1.4-2.8 kc noise. The CI for 2.8-5.6 kc noise (31) has a small but significant loading; this occurs because of the correlations of 0.32 and 0.35 between the two measures of CI (Table IV). Notice that this factor does not include the TTS from the 2.8-kc tone (15).

$F_c$ : high-frequency TTS. The main component of this factor comes from the TTS produced by 2.8-5.6 kc noise (16) and of course the CI based on this data (31). In addition, the average resting threshold at 2.8, 4, and 5.6 kc is involved in the determination of this factor. It is interesting to note that the CI for impulse noise (17) is also implicated here, and so does not have a factor all its own, despite only negligible raw correlations between 16 and 17 (Table IV). Curiously, the impulse CI has a positive loading, implying that the ears showing the most TTS from 2800-5600 cps noise will show the least from impulses. This is the only factor upon which the recovery rate (25) shows a significant loading, and only weakly at that.

$F_d$ : sex. This factor is determined mainly by sex (21). Since the difference in ear projection between males and females was pronounced, this variable (22) is also strongly involved. Two measures with significant loadings, though in opposite directions, are 1 and 16. This is related to the fact that the men showed significantly more TTS than the women at low frequencies, but less after the highest-frequency exposure.

$F_e$ : threshold. All the threshold measurements (27-30) are found in this factor, indicating that ears tending to be low in sensitivity at one frequency are likely to be low at others also. Variable 26, Békésy excursion, has a negative loading on this factor, which suggests that the listeners with the largest Békésy spread—that is, the "worst" subjects—tended to show the lowest thresholds. One possible interpretation of this fact is that perhaps such persons push the voting button as soon as they hear the pulsed test tone, just as they are instructed, but release it only after missing three or four pulses; this would produce a record indicating a spuriously high sensitivity.

$F_f$ : adaptation. Only two variables had significant loadings on this factor for both RE and LE analyses: the magnitude of the adaptation at 2.7 minutes (19) and the change in adaptation from 0.7 to 4.4 minutes (20), as measured in Week 20. The loadings for the two are in opposite

directions: those listeners showing the least adaptation halfway through the 5-minute exposure showed the greatest change from the first measurement to the third. Clearly, adaptation of this sort has no consistent relation to TTS. In fact, it seems, both from the factor analysis and the raw correlation matrix, to be completely unrelated to any other aspect of the auditory mechanism. This result stands in opposition to Tanner's (1955) claim that there is a negative correlation between adaptation and fatigue. There appear to be only two fundamental differences between Tanner's experiment and the present one, since the adapting exposure was the same. First, an interrupted tone was used here, in order to minimize the effect of adaptation in the control ear during the adjustment (Wright, 1960), while Tanner no doubt used a continuous one. The second difference lies in the specific instructions used; for instance, great stress was laid in the present instance on bracketing the final setting. Tanner's instructions are not given. Which of these possibilities is responsible for the difference in outcome remains to be determined.

*F<sub>p</sub>: "distortion."* This factor throws together the TTS produced by 15-minute earphone exposures to 1.4-2.8 kc noise (both continuous and intermittent) and "distortion"—the masking of a contralateral 6.7-kc test tone by a 0.7-1.4 kc noise. The loadings for the TTS and the masking are in opposite directions: the ears showing the most TTS are those displaying the least contralateral masking. This negative correlation between TTS and both kinds of contralateral remote masking (see also correlations in Table IV between 23 and both 2 and 5) was most surprising. If a high TTS means, other things being equal, a higher effective energy reaching the cochlea, then one might expect more distortion and a positive correlation. Apparently this is not the case; the actual results may demonstrate that if the cochlea scatters a great deal of energy to remote frequencies, as implied by a large amount of contralateral remote masking, then there is perforce somewhat less energy available at the exposure frequency to produce TTS.

*F<sub>h</sub>: low-level TTS.* This factor depends almost entirely on the TTS produced by test tones of 2, 4, and 8 kc at low intensities (18). There is a significant loading also for the otherwise-anomalous 3-minute exposure to a 2.8-kc tone at 116 db SPL. It may be, therefore, that this factor should be termed "high-frequency pure-tone TTS."

Such were the 8 factors that emerged from a varimax analysis of these particular 32 variables. In addition, the RE analysis had a ninth factor on which there was a loading of 0.77 for the TTS produced by the step exposures to 1.4-2.8 kc noise (9) and of -0.67 for the low-frequency CRM (23). Again, this finding probably indicates that if the energy of a stimulus is scattered to remote frequencies, it can do less mischief at its nominal frequency.

## V. Discussion

There is, then, limited support for the viewpoints of both the general- and the specific-susceptibility hypotheses. Although a principal factor based mainly on the TTS from all exposures to stimuli below 2 kc can be found in the present particular set of data, only 33% of the variance explainable by ten different factors can be thrown into this single factor no matter what rotation of axes are made in the analysis. As the variance estimates at the bottom of Table VI show, the most salient factor in the varimax analysis (in which an attempt is made to maximize loadings on all factors) accounts for 24% of the variance and so is nearly as potent as the first factor in the principal-factor analysis.

One must remember, too, that to a certain extent the dice were loaded to produce a fairly salient principal factor. As Webster has put it, "the same auditory test groups itself with different bedfellows depending upon the choice of bedfellows it has to choose from" (Webster, 1964). In the present instance, there were a great many tests involving the mid-frequency range, but only one each, for example, using 0.35-0.7 and 2.8-5.6 kc noise. Thus it is inevitable that unless *each* of the tests had been truly completely independent, there would emerge a common factor involving the mid-frequency exposures.

### A. The Aural Reflex

On the other hand, there was no necessity for *all* the stimuli involving frequencies at and below 2 kc to be grouped together in the results. The fact that they were suggests that the strength of the middle-ear muscles may be the underlying physiological variable responsible, since it seems likely (see Jepsen, 1963, for a brief review of the evidence) that contraction of these muscles affects the transmission of frequencies only below 2 kc. Perhaps the greater average resistance of the females to TTS from low-frequency exposures means that they have stronger middle-ear muscles or that they are more capable of sustained contraction over a period of several minutes.

### B. A "Best" Susceptibility Test?

The present research shows conclusively that there is little correlation between the various susceptibility tests heretofore proposed, just as Greisen (1951) found. A similar result has recently been reported by Fletcher and Loeb (1965). Taking at face value the relative loadings of the different variables on the results of the varimax analysis, we should apparently measure, in order to determine as many facets as possible of the susceptibility of an ear to TTS, the following:

- (1) the average TTS<sub>2</sub> produced at 1.7, 2, and 2.4 kc by a 3-minute monaural exposure to 0.7-1.4 kc noise at 120 db;
- (2) the CI for 1.4-2.8 kc noise—i.e., the SPL of a 1-minute monaural exposure that will just produce a TTS<sub>0.5</sub> of 10 db at 4 kc;
- (3) the TTS<sub>2</sub> at 5.6, 6.7, and 8 kc produced by successive 1-minute monaural exposures at increasing levels (through 125 db SPL) to 2.8-5.6 kc noise;
- (4) the distance the ears project from the head;
- (5) the TTS<sub>2</sub> at 2.8, 4, and 5.6 kc produced by a 15-minute binaural exposure to 1.4-2.8 kc noise at 106 db SPL; and
- (6) the resting threshold at 1 and 1.4 kc.

The analysis also implies that one should measure the rate of change of adaptation (peristimulatory fatigue), and the TTS produced by low-SPL exposure, but one would do so only if he believed that these would have a significant predictive value in regard to permanent threshold shifts. I do not.

Notice that there is no necessity to separate tests using pure tones from those using noises, earphone versus field exposures, or steady versus intermittent noise, according to the factor analysis.

In essence, we should measure TTS at three separate frequency regions for 3-minute monaural and 15-minute binaural exposures. However, there is evidence that the three frequency regions need not be sampled in separate tests, but that a single exposure to a composite noise can be used to produce TTS in all three ranges. The TTS produced by the composite noise of Week 21 apparently samples both factors  $F_a$  and  $F_b$  (Table VI): the TTS<sub>1.3</sub> at 1, 1.4, and 2 kc (6) has a loading of 0.73 on factor A despite the fact that the mean value of the TTS was only 4.4 db. Similarly, the TTS<sub>2.2</sub> at 2.8, 4, and 5.6 kc (10) has a loading of 0.62 on factor  $F_b$ , which is nearly as high as that of any other TTS measurement (in factor  $F_b$ , 9 through 14). Even if a different *absolute* value of TTS at a given frequency is produced when the noise primarily responsible for its generation is joined by noises of other frequencies, the *relative* rank order of listeners in regard to the TTS produced is apparently unchanged.

The best single test for susceptibility to TTS in general, then, would appear to be a noise whose composition is such that it will produce moderate shifts at frequencies from 1.5 to 8 kc. Whether, as far as prediction of susceptibility to permanent loss is concerned, this should be a 3-minute monaural exposure, a 15-minute binaural exposure, or perhaps an even longer one, is yet to be determined. In addition, if a particular industrial noise is unusually complicated (spectrally or temporally), or has an impulsive element, it seems inescapable that the best estimate of

susceptibility to permanent damage will be provided by a TTS test using *that specific noise*.

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#### REFERENCES

- Christiansen, E. (1956). *Acta Oto-Laryngol.* 46, 99.  
 Epstein, A., Katz, J., and Dickinson, J. T. (1962). *Acta Oto-Laryngol.* 55, 81.  
 Falconnet, P., Portmann, M., and Alavoine, J. (1955). *Ann. Oto-Laryngol.* 72, 747.  
 Fletcher, J. L., and Loeb, M. (1965). *J. Auditory Res.* 5, 41.  
 Fletcher, J. L., and Riopelle, A. J. (1960). *J. Acoust. Soc. Am.* 32, 401.  
 Flügel, J. C. (1920). *Brit. J. Psychol.* 11, 105.  
 Fosbrooke, J. (1830-1831). *Lancet* 1, 740.  
 Gallagher, J. B., and Goodwin, J. E. (1952). *Arch. Ind. Hyg. Occupational Med.* 6, 332.  
 Graessner, L. (1951). *Acta Oto-Laryngol.* 39, 132.  
 Harman, H. H. (1960). "Modern Factor Analysis." Univ. of Chicago Press, Chicago, Illinois.  
 Harris, J. D. (1954). *Laryngoscope* 64, 89.  
 Jepsen, O. (1963). In "Modern Developments in Audiology" (J. Jerger, ed.), pp. 194-237. Academic Press, New York.  
 Jerger, J. F., and Carhart, R. (1955). *U. S. Air Force School Aviation Med. (San Antonio, Texas) Rept.* 55-64.  
 Jerger, J. F., and Carhart, R. (1956). *J. Acoust. Soc. Am.* 28, 611.  
 Katz, J. (1965). *J. Acoust. Soc. Am.* 37, 923.  
 Link, R., and Handl, K. (1955). *Arch. Ohren-Nasen-Kehlkopfheilk. Ver. Z. Hals-Nasen-Ohrenheilk.* 167, 610.  
 Miller, J. D., Watson, C. S., and Covell, W. P. (1963). *Acta Oto-Laryngol. Suppl.* 176.  
 Palva, T. (1958). *Arch. Otolaryngol.* 67, 228.  
 Peyser, A. (1930). *Deut. Med. Wochschr.* 56, 150.  
 Peyser, A. (1940). *Acta Oto-Laryngol.* 28, 443.  
 Peyser, A. (1943). *Acta Oto-Laryngol.* 31, 351.  
 Reid, C. (1946). *J. Laryngol. Otol.* 61, 609.  
 Rüdel, L. (1954). *Acta Oto-Laryngol.* 44, 502.  
 Small, A. M., Jr. (1963). In "Modern Developments in Audiology" (J. Jerger, ed.), pp. 287-336. Academic Press, New York.  
 Tanner, K. (1955). *Acta Oto-Laryngol.* 45, 65.

- Theilgaard, E. (1949). *Acta Oto-Laryngol.* 37, 347.
- Theilgaard, E. (1951). *Acta Oto-Laryngol.* 39, 525.
- Urbantschitsch, V. (1981). *Arch. Ges. Physiol. Pfluegers* 24, 574.
- van Dishoeck, H. A. E. (1956). *Muench. Med. Wochschr.* 98, 1625.
- Ward, W. D. (1961). *J. Acoust. Soc. Am.* 33, 1034.
- Ward, W. D. (1962a). *J. Acoust. Soc. Am.* 34, 234.
- Ward, W. D. (1962b). *J. Acoust. Soc. Am.* 34, 1610.
- Ward, W. D. (1964). *Acta Oto-Laryngol.* 58, 139.
- Ward, W. D. (1965a). *J. Acoust. Soc. Am.* 38, 121.
- Ward, W. D. (1965b). *J. Occupational Med.* 7, 595.
- Ward, W. D., Selters, W., and Glorig, A. (1961). *J. Acoust. Soc. Am.* 33, 781.
- Webster, J. C. (1964). *J. Speech Hearing Res.* 7, 292.
- Wheeler, D. E. (1950). *Arch. Otolaryngol.* 51, 344.
- Williams, R. J. (1956). "Biochemical Individuality; The Basis for the Genetotrophic Concept." Wiley, New York.
- Wilson, W. H. (1943). *Arch. Otolaryngol.* 37, 757.
- Wilson, W. H. (1944). *Arch. Otolaryngol.* 40, 52.
- Wright, H. N. (1960). *J. Acoust. Soc. Am.* 32, 1558.